

Christchurch Medical Research Society

Scientific Meeting

Friday 25 July 2003

12:00 – 1:30 pm in the Rolleston Lecture Theatre,
Christchurch School of Medicine & Health Sciences

12:00 POSTER RECYCLING SESSION and LUNCH in the ground floor foyer. *The posters will be on display from the afternoon of Wed 23 July: take a look at your leisure in the days before and discuss the findings with the author over lunch.*

12:30 ORAL PRESENTATIONS

12:30 **Christchurch hospital's chest pain assessment unit: the first six months**

Calum M Young*, Ian G Crozier, C Cruickshank, H Ikram

12:45 **The association between chronic pain and psychosocial functioning following spinal cord injury**

Mark A Turner*

1:00 **A survey of colonoscopic surveillance for dysplasia in patients with inflammatory bowel disease**

Richard B Geary*, Christopher J Wakeman, Murray L Barclay, Bruce A Chapman, Michael J Burt, Judith A Collett, Frank A Frizelle

1:15 **Urocortin-1 Infusion in Normal Humans**

Mark E Davis*, Chris J Pemberton, Timothy G Yandle, John G Lainchbury, Miriam T Rademaker, M Gary Nicholls, Christopher M Frampton, A Mark Richards

* Contestants for the CMRS Young Researcher Prize

Christchurch hospital's chest pain assessment unit: the first six months

Calum M Young, Ian G Crozier, C Cruickshank, H Ikram

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A Chest Pain Assessment Unit (CPAU) was established at Christchurch Hospital in November 2001 to assess patients with acute chest pain who had no high risk features at presentation. The Unit aimed to reduce length of stay to less than 24 hours and to minimise incorrect diagnosis. Prospective audit was undertaken of the first six months.

Patients admitted to CPAU have myocardial infarction excluded by troponin testing, with most then having pre-discharge exercise treadmill test.

232 patients (122 male, mean age 53 years) were assessed in CPAU. 6 patients (2.6%) had non-ST elevation myocardial infarction. Exercise treadmill testing was performed in 197 patients (84.9% of all CPAU patients), with 157 (79.7%) being negative.

172 patients (74.1%) were discharged directly home from CPAU, with median length of stay 21.0 hours. The remaining 60 patients were admitted to the ward for further management. Median length of stay for this group was 15.2 hours in CPAU and further 43.5 hours on the ward.

Overall, a final diagnosis of acute coronary syndrome was made in 21 patients (9.1% of all CPAU patients) of which 17 had angiographically proven disease. In six month follow-up of the patients discharged from CPAU without a diagnosis of acute coronary syndrome there were three readmissions with non-cardiac chest pain, one readmission with a non ST-elevation myocardial infarction, and no known deaths due to coronary disease. We conclude that CPAU allows for efficient and safe assessment of patients with chest pain who do not have high risk features at presentation.

The association between chronic pain and psychosocial functioning following spinal cord injury

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The aims of the present study are to examine the prevalence of chronic pain secondary to spinal cord injury and to examine the extent chronic pain impacts on a range of indicators of psychosocial functioning.

A cross-sectional survey, in which data is gathered by face-to-face interview, was conducted. The cohort consists of 122 individuals (89% of those meeting eligibility criteria) with traumatic SCI and residual neurological impairment who were resident in Christchurch at the time of injury.

Over three-quarters (77%) of participants reported the presence of some degree of chronic pain. After controlling for a range of demographic, injury-related, and personality, social, and cognitive factors, increasing levels of characteristic pain intensity were found to be significantly associated with a range of indicators of psychosocial functioning. Those with high characteristic pain intensity had increased rates of psychiatric morbidity (compared to those with no pain) including: high psychological distress (OR = 7.88; 95% CI 2.05-30.28), suicidal ideation (OR = 4.26; 95% CI 1.20-15.08), major depression (OR = 22.18; 95% CI 3.08-159.92), anxiety disorders (OR = 7.60; 95% CI 1.21-47.59), and overall psychiatric morbidity (OR = 4.35; 95% CI 1.27-14.95). They also had increased rates of poor social functioning including: small social network (OR = 4.96; 95% CI 1.64-14.99), low social participation (OR = 4.65; 95% CI 1.30-16.55), and low social integration (OR = 7.60; 95% CI 1.55-37.17). Finally, after adjustment for confounding, higher levels of characteristic pain intensity are associated with poorer adjustment to disability and lower life satisfaction.

Chronic pain places a significant additional burden on psychosocial functioning. Identification of specific areas of psychosocial functioning associated with higher characteristic pain intensity should help rehabilitation professionals target interventions to reduce the suffering associated with SCI pain.

A survey of colonoscopic surveillance for dysplasia in patients with inflammatory bowel disease

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Patients with inflammatory bowel disease have an increased risk of developing colorectal cancer. Screening colonoscopy is practised to identify dysplasia, allowing early colectomy and reduction in colorectal cancer incidence and mortality.

The aim of this survey was to determine the pattern of screening for dysplasia by colonoscopy in New Zealand.

A survey was developed and posted to all gastroenterologists and general and colorectal surgeons in New Zealand. Doctors were identified using a variety of professional registries. Of 196 posted surveys, there were 156 replies of which 120 were suitable for analysis

The definition of dysplasia was thought to be “unequivocal neoplastic change” by 21% of respondents and “preneoplastic change” by 68%. Most doctors commenced surveillance colonoscopy ten years after the onset of symptoms and most perform colonoscopy every three years. The mean number of biopsies was 19 (range 3-42). Recommendations following the finding of Low Grade Dysplasia (LGD) were variable with 18% of doctors referring this group for colectomy. Seventy-six percent of doctors recommended colectomy if High Grade Dysplasia (HGD) was found. Ninety-six percent of respondents recommend colectomy for Dysplasia Associated Mass Lesions. Twenty percent of doctors stated access to endoscopy delayed surveillance colonoscopy.

There were significant differences between the practice of different groups of doctors, especially between general surgeons and other respondents. The results show a wide variety of colorectal dysplasia screening practice in New Zealand. While most doctors recognise the significance of HGD, LGD remains suboptimally treated. This may be due to confusion concerning the definition of dysplasia.

Urocortin-1 Infusion in Normal Humans

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Urocortin-1, a member of the corticotropin-releasing factor family, has been shown in animal studies to have effects on the pituitary-adrenal axis, the cardiovascular system, circulating neurohormones, renal function and to suppress appetite. For the first time in humans we have evaluated such effects of infused urocortin-1 as well as actions on plasma ghrelin, a hormone known to increase appetite. We also assessed urocortin-1 pharmacokinetics.

Eight healthy male volunteers taking a diet of constant sodium and potassium content received 50 µg of urocortin-1 intravenously over one hour in a placebo-controlled, randomized, time-matched, cross-over study.

Urocortin-1 infusion compared to placebo increased plasma levels of corticotropin (9.5 ± 1.7 vs 4.2 ± 0.7 , $p < 0.001$), cortisol (432 ± 43 vs 213 ± 40 , $p < 0.001$) and atrial natriuretic peptide (ANP) (8.5 ± 1.1 vs 6.9 ± 0.7 , $p = 0.019$), whilst suppressing plasma ghrelin ($p = 0.008$). No hemodynamic or renal effects were observed at the dose used. The plasma urocortin-1 $t_{1/2}$ was 52 minutes based on a one compartment model.

In conclusion, a brief intravenous infusion of 50 µg of urocortin-1 stimulates plasma ACTH, cortisol and ANP secretion and suppresses plasma ghrelin in healthy male volunteers. The latter effect might contribute to the anorexic action of urocortin-1.